Cerebral Vasculature and Strokes

Introduction

You saw how tracts and nuclei relate to blood vessels in the cortex. There, we only dealt with the anterior and middle cerebral arteries and how they related to the different tracts. We also pointed out a few important features, like Broca's area. Now, we are going to study the cortex from the perspective of the blood vessels. This lesson is about learning the major arteries of the cortex and exploring the lobes of the cortex—what they do and what happens when there is a stroke and they are lost.

Because the brain is a three-dimensional structure, it isn't easy to explain in words what the arteries irrigate—but it is easy to represent in illustrations. We will both provide the words (which tend to be effortful to interpret) and the illustrations (which tend to make things easy).

The goal is not to learn all the arteries of the brain. The goal is to learn the major arteries of the circle of Willis and the territories of the three main arteries of the cerebrum, as well as to be able to identify strokes of those major branches. Most strokes do NOT involve these major branches. Rather, they involve a distal branch of a main artery. It becomes too dense to memorize the symptoms of their branches, and because you will obtain imaging on a patient who presents with a focal neurologic deficit, you won't have to infer the causal artery. Therefore, we're going to use this lesson both to teach you about atherosclerosis of the brain (which is the same affliction as in the Cardiology module, only now in the vasculature of the brain) and to reinforce the primary function of each lobe, using strokes to tell that story.

Cerebral Vasculature and Cerebral Strokes (vs. Brainstem)

In this curriculum, we have opted to portray strokes of the brain and brainstem as exactly those—cortical strokes (frontal, parietal, temporal, and posterior lobe strokes) and brainstem strokes (midbrain, pons, medulla, cerebellum). This requires stating aloud because of the posterior cerebral arteries. From a vascular perspective, the **anterior circulation** consists of the middle cerebral arteries and the anterior cerebral arteries derived from the two internal carotid arteries. In contrast, the **posterior circulation** comprises the posterior cerebral arteries, which branches from the basilar artery, which comes from the vertebral arteries. The anterior circulation is connected to the posterior circulation by posterior communicating branches between the middle and posterior cerebral arteries. Those anastomoses are small, an indication that the middle cerebral artery is not derived from the same vascular trees as the posterior cerebral.

But there is a literal separation of the cortex from the brainstem. Sure, they come together at the basal ganglia and deep brain, but the tentorium cerebelli is a firm membrane that keeps the cerebellum quite physically distinct from the posterior cortex. And although the posterior cerebral artery is derived from the posterior circulation, it perfuses the cerebrum, not brainstem

Because (conveniently) there are anterior, middle, and posterior stroke syndromes—each accounting for one of the three main vessels that perfuse the cerebrum—we have opted to teach **cerebral strokes** as one subject and **brainstem strokes** as another. Intellectually, note that they are intimately connected because they are so close together—the circle of Willis is only one centimeter in diameter. As a memory tool, mentally separate cerebral strokes from brainstem strokes.

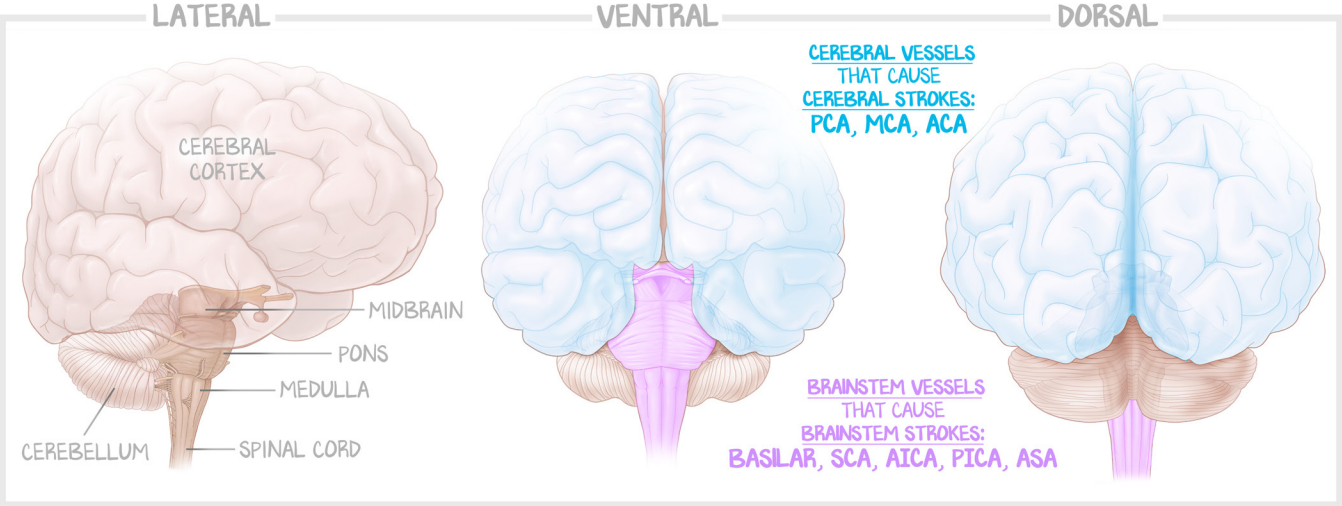

Figure 4.1: Functional and Anatomic Circulations
The lateral view shows the brain, brainstem, and cerebellum as a connected unit—the anatomic truth. Although we are about to show you that the posterior cerebral arteries are really part of the posterior circulation, we want you to think a little differently. Occlusions of the cerebral vessels cause cerebral strokes, whereas occlusions of the brainstem vessels cause brainstem strokes. The cerebrum is perfused by the anterior, middle, and posterior cerebral arteries; the brainstem is perfused by the basilar, superior cerebellar artery (SCA), and the AICA, PICA, and ASA, which you'll see in the lesson on brainstem strokes.

Circle of Willis

The circle of Willis is located in the **midbrain**. It is an anastomosis between three anatomic arterial origins—the left internal carotid artery, the right internal carotid artery, and one basilar artery.

Each **internal carotid artery** passes into the temporal bone through its respective carotid foramen (the hole in the bone) into the carotid canal (the hole through the bone). Each then exits into the cranium through the cavernous sinus, emerging just underneath the optic chiasm, lateral to the sella turcica. The two internal carotids are met by the single **basilar artery**, which runs anteriorly over the medulla and pons. As the basilar artery approaches the midbrain, just under the thalamus, it branches into two **posterior cerebral arteries** (PCA). Each internal carotid passes back an anastomotic connection to its associated PCA, a **posterior communicating artery** (Pcom) for each side.

Each internal carotid branches into two arteries—the **middle cerebral artery** (MCA) and the **anterior cerebral artery** (ACA). Technically, the internal carotid continues as the MCA, but it is easier to think of it as a branch. The ACAs complete the circle of Willis by connecting to each other with a single **anterior communicating artery** (Acom).

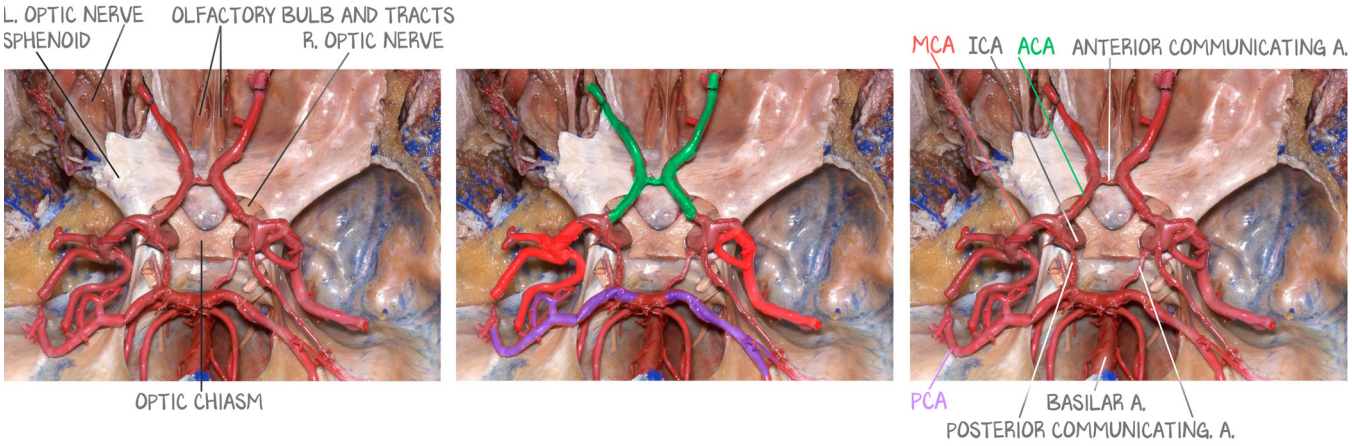

Figure 4.2: Circle of Willis
This prosection of the circle of Willis is used merely to demonstrate what the circle is and where it comes from. The first panel points out adjacent landmarks, while the second identifies the vessels. Only half of the second panel is labeled so that you can see an unobstructed view of the contralateral side. The final panel identifies the six major vessels (three on each side) that define each of the major vascular territories—the anterior (green), middle (red), and posterior (purple) arteries.

We want you to see the brain, all of the cortex, not based on the anatomic correctness of the vasculature, but from the anatomic correctness of the dura. The entire cortex is separated from the brainstem by the tentorium cerebelli. This strategy isolates objects in your memory—the cortex has these vessels and symptoms, whereas the brainstem has those vessels and symptoms. The circle of Willis is at the base. This gives us **a left-right mirror** but no anterior-posterior mirror. Be careful; there is no anterior-posterior mirror! Although, if you consider the cerebrum, there is obviously no anterior-posterior mirror, but there is a left-right mirror. The vascular supply mimics the symmetry of the tissue it perfuses.

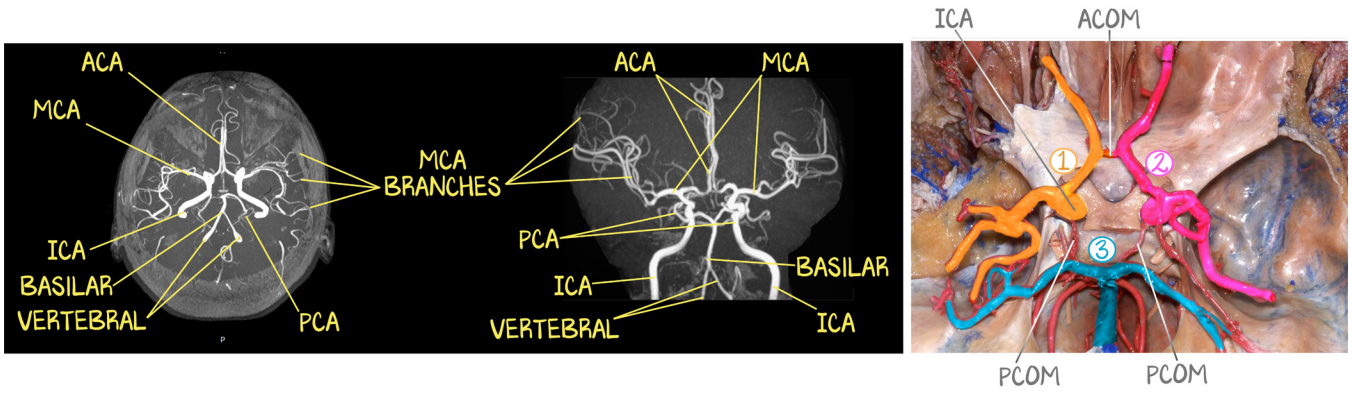

Figure 4.3: Where the Circle of Willis Comes From
The circle of Willis is built from three independent but connected circulations, as shown by the color-enhanced prosection on the right. The left internal carotid artery gives rise to the left middle cerebral and left anterior cerebral arteries, the right internal carotid artery gives rise to the right middle cerebral and right anterior cerebral arteries, and the basilar artery gives rise to two posterior cerebral arteries. The three circulations are connected by communicating arteries—two posterior communicating arteries connect each internal carotid territory with the basilar, and an anterior communicating artery connects the two internal carotid territories. Using the angiograph, you can trace the origins of each of the three circulations. The posterior circulation is derived from vertebral arteries, which merge to form the basilar artery. The anterior circulations are each derived from a large internal carotid artery.

Cortical Distribution of the Main Arteries

Mentally, cleave off the basilar artery and everything below. We focus now on three arteries: the anterior, middle, and posterior cerebral arteries. Every lobe has a shared arterial distribution—some portion of each lobe is perfused by two arteries. Because the brain is a three-dimensional structure, you must consider the distribution in three dimensions. This is usually depicted with a medial sagittal view (from the corpus callosum looking out), a lateral sagittal view (from the skull looking in), and a superior transverse view (top looking down). We are going to show you oblique views so you can see the relative relationships and describe them with tedious accuracy. As long as you can close your eyes and see which artery covers what, you're good.

The left and right arteries perfuse the left and right hemispheres independently. The left ACA, MCA, and PCA perfuse the left hemisphere and none of the right. The right ACA, MCA, and PCA perfuse the right hemisphere and none of the left. Each artery perfuses its own territory **without anastomosis or overlap**. This means that the most distal cortex is a **watershed area**, the most vulnerable to reduced perfusion. The major watershed areas are at the ACA-MCA territories and the MCA-PCA territories. We are going to speak in the singular for clarity, and you know that whatever is said for one hemisphere is true for the other.

The **anterior cerebral artery** is the "*medial superior artery*." It perfuses its half of the corpus callosum, all of the medial aspect of the frontal lobe, the anterior of the frontal lobe, most of the medial parietal lobe, and the most superior aspect of the lateral frontal lobe. It extends anteriorly from the circle of Willis in a very medial position (both ACAs are near each other), ascends along the corpus callosum, and extends posteriorly over the top of the corpus callosum. The cerebral falx prevents the two ACAs from ever meeting again. The ACA lines the corpus callosum. It is able to perfuse its territory because its branches reach out in front, reach up onto the lateral cortex, and reach back to the parietal lobe.

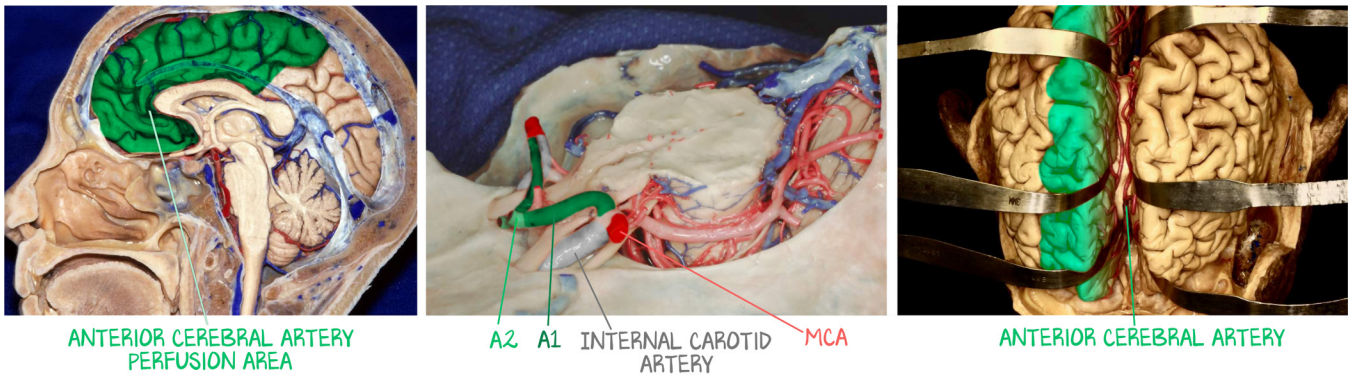

Figure 4.4: The Anterior Cerebral Artery
The anterior cerebral artery exits the circle of Willis anteriorly, connected to its partner by the anterior connecting artery. You can follow its main branch on both the sagittal and superior medial sections. It hugs the corpus callosum and extends branches upward. The vascular supply is highlighted in green.

The **middle cerebral artery** is the "*lateral artery*." It is derived from the internal carotid and runs nearly perpendicular to the ACA. It runs **within the Sylvian fissure**. The circle of Willis is at the base of the brain. The MCA runs laterally under the frontal lobe, wraps around the frontal lobe into the Sylvian fissure (now in between the temporal and frontal lobes), then courses posteriorly towards the occipital lobe. It quickly branches into superior and inferior terminal branches. The **superior** branch reaches up, out of the Sylvian fissure onto the lateral side of the brain—the frontal and parietal lobes. The **inferior** reaches **up** out of the fissure, and so irrigates the medial (inside) of the temporal lobe, the anterior, and superior half of the temporal lobe.

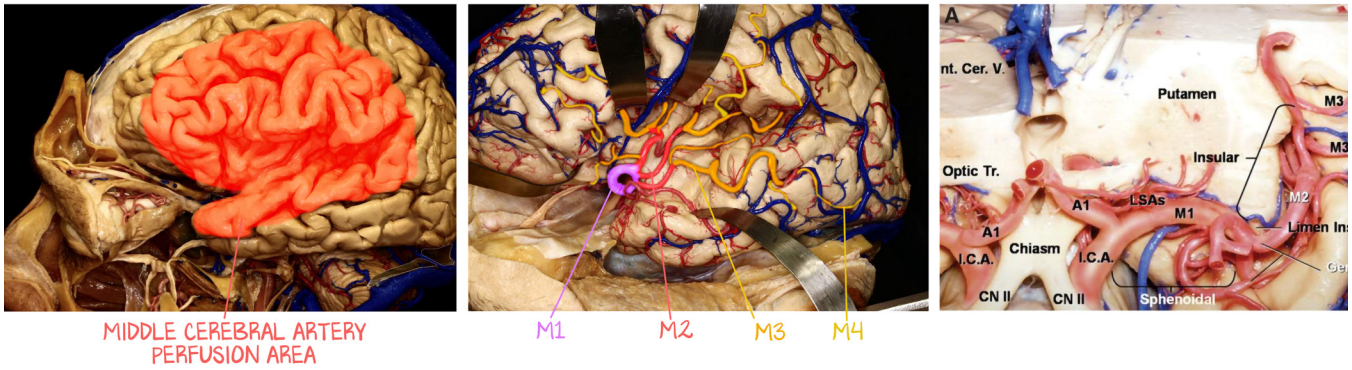

Figure 4.5: The Middle Cerebral Artery
The middle cerebral artery exits the circle of Willis and travels laterally. The retracted lateral view has been colorized to follow the different segments. You are not to learn the names of the branches or what M1–M4 mean, merely use the colorization to follow its branches and see how it's main path is within the Sylvian fissure, spreading outward to perfuse the lateral frontal lobe, lateral parietal lobe, and medial temporal lobe.

The **posterior cerebral artery** comes off the back of the circle of Willis from its branching off the basilar artery and courses towards the occiput around the cerebral peduncle, over the tentorium cerebelli, and under the temporal and occipital lobes. It is going to get the rest of the cortex that isn't covered by the ACA and MCA—specifically, the visual cortex of the occipital lobe, the rest of the occipital lobe, the lower half of the lateral temporal lobe, and most of the medial temporal lobe. The deep brain is shared between the MCA and PCA.

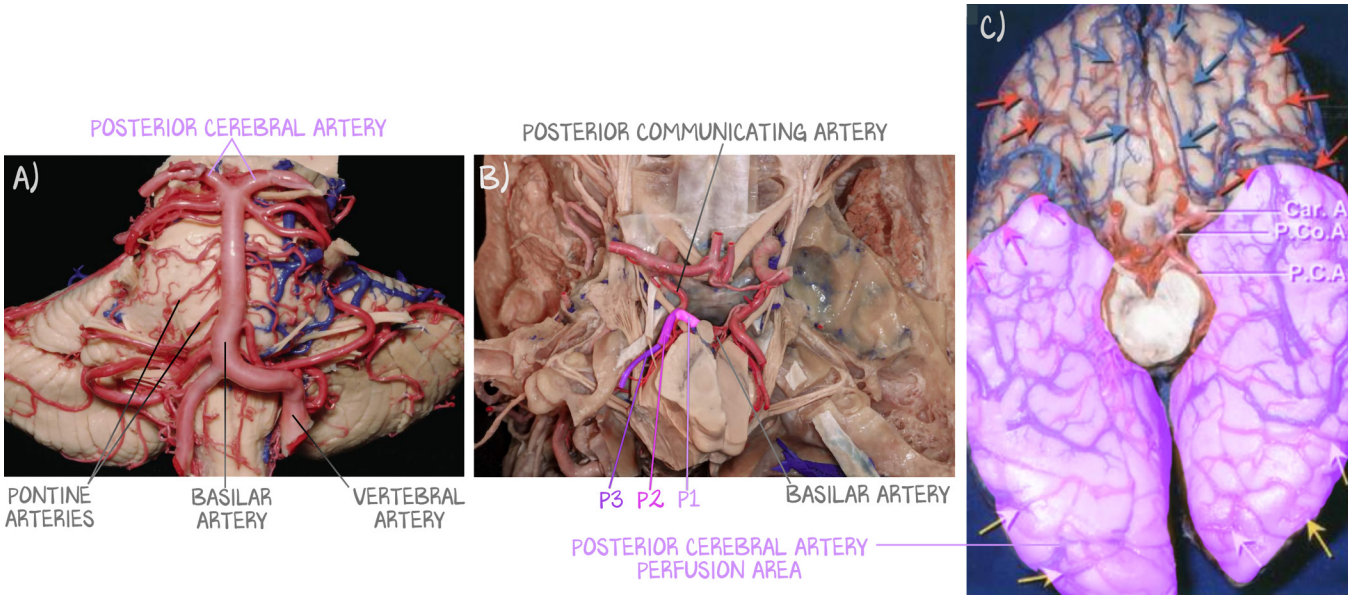

Figure 4.6: The Posterior Cerebral Artery
(a) The basilar artery ascending the anterior brainstem, the cerebellum on either side of it. The only point of this perspective is to show you that the posterior cerebral arteries are a bifurcation of the basilar artery. (b) Multi-planar dissection showing the artery of Willis, with one posterior cerebral artery labeled. (c) The territory of the posterior cerebral artery is the occipital lobes, posterior parietal, and any of the temporal lobe missed by the MCA.

Types of Strokes

Strokes are divided into **ischemic** and **hemorrhagic**. They both cause damage to the brain parenchyma, so they are both strokes. But one is because there is bleeding—an open blood vessel leaking blood (which we covered in Neuroscience: Cortex #4: *ICP, Cerebral Edema and Brain Bleeds* and will not cover here)—and the other is because of occlusion of a vessel, leading to ischemia or infarction.

Within ischemic stroke, there are three overarching etiologies: thrombotic, embolic, and lacunar. We can pair two with the pathophysiology you already know. Lacunar is hyaline arteriolosclerosis (hypertensive), thrombotic is atherosclerosis.

Lacunar. Hyaline arteriolosclerosis is the histologic finding of microvascular disease associated with leaky capillaries. Capillaries get leaky due to hypertension, and the process is accelerated by diabetes. Hyaline arteriolosclerosis affects arterioles (arteriole-o-sclerosis)—small blood vessels. These changes result in the formation of **small cavitary infarcts**, called lacunae. They tend to form in the deep brain—internal capsule, thalamus, caudate—and the pons. They are small, and so may be silent (asymptomatic). The more of them there are, the more damage is done. Patients may develop **cognitive decline** and never know they have them. Because hyaline arteriolosclerosis occurs in "career hypertension," these patients are also at increased risk for intraparenchymal hemorrhage. Don't be fooled; the associated buzzword for this diagnosis is **lipohyalinosis**, which is "hyaline arteriolosclerosis" by a different name. These occur in the **lenticulostriate arteries**, branches of the MCA that perfuse the internal capsule, thalamus, and basal ganglia. These are the strokes that can cause **devastating hemiparesis and hemisensory loss**, given their relation to the internal capsule. One lacunar infarct won't present with symptoms. Many lacunar infarcts in the right place, will. See the final section for details.

Atherosclerosis. A brief review of the pathogenesis of atherosclerosis is provided here. It is brief, as we expect you to have learned this in the Cardiac module. Skip ahead if you remember it. Atherosclerosis affects the large-to-medium elastic arteries, almost always at **branch points** (here, that means the ACA, MCA, and PCA). It is caused by LDL deposition under the endothelial cells (**lipid core**). Macrophages try to digest the LDL, fail, and die in place, creating a **necrotic lipid core**. Vascular smooth muscle cells of the tunica media proliferate and migrate over the necrotic core to create a **fibrous cap**. The cap keeps the thrombogenic necrotic core from the bloodstream but doesn't prevent more LDL from adding to the core. Over time, the atherosclerotic plaque (necrotic core with fibrous cap) grows into the lumen. At critical stenosis (≥ 70%), symptoms will present themselves. If the demand of the organ goes up, there will be ischemia. If the demand then falls, the ischemia is alleviated. Whereas exercise stresses the heart and eating stresses the bowel, nothing really alters the brain's demand. Therefore, most atherosclerosis in the brain continues to worsen until the plaque is larger than merely critical stenosis; it advances undetected until it becomes an unstable plaque. An unstable plaque can rupture and thrombose. **Rupture and thrombosis** may lead to a **transient ischemic attack** (TIA, the NSTEMI equivalent in the brain) or **cerebrovascular accident** (100% occlusion, the STEMI equivalent in the brain). Because atherosclerosis affects large-to-medium-sized vessels, the vascular territory is usually large, and the symptoms are overt. This is the stroke we're going to be talking about moving forward.

Embolic. Embolic strokes are caused either by **atrial fibrillation** (atrial appendage clot, the reason primary stroke prophylaxis is given to patients with AFib) or by **atherosclerosis, rupture and thrombosis, then embolism somewhere else**, most commonly the **carotid arteries**. Carotid artery stenosis is atherosclerosis of the carotid arteries. Carotid artery stenosis can reach nearly 100% occlusion without symptoms because of the anastomosis of the two carotids through the circle of Willis. If the stenosis is bad enough, the patient may present with stroke symptoms, usually in the form of a TIA. But these are unstable plaques, and can also acutely thrombose. Again, with collateral circulation, the thrombosis isn't experienced by the patient. But if that thrombosis then **embolizes**, it will do so downstream, where the arteries get progressively smaller. Embolic, like thrombotic, tends to affect the

large-to-medium arteries (ACA, MCA, PCA). Embolic and thrombotic strokes are essentially the same disease and are treated the same way (at least acutely). There are some variations when it comes to AFib atrial appendage (warfarin or NOAC when these are not indicated in atherosclerotic embolism) and a patent foramen ovale (paradoxical embolus), but those are considerations for Cardiology during the clinical sciences. The point is that both embolic and thrombotic strokes present with **overt focal neurologic deficits.** Embolic stroke is why part of the work-up for a stroke involves carotid ultrasound (assessing for carotid stenosis) and echocardiogram (assessing for atrial appendage clot). It is also why we told the story of embolic stroke using carotid artery stenosis and AFib with atrial appendage clot.

TIA and CVA

A TIA is an episode of **reversible** neurologic dysfunction. It is caused by **focal ischemia** (acute rupture and thrombosis of a plaque) that goes away on its own. There must also be the **absence of acute infarction**, as evidenced by MRI, and no lasting neurologic deficit. This is a technical definition that was established in 2010. Clinical practice typically does not perform MRI on everyone with focal neurologic deficits that are transient and resolve spontaneously. It does NOT mean "get an MRI on everyone in whom you consider TIA as the diagnosis." But if you make the diagnosis of TIA and there are vascular risk factors, that person needs an MRI soon. As in today or in the next few.

A **stroke** is an umbrella term that means "anything that causes acute focal neurologic deficit." A **cerebrovascular accident** (CVA) usually means **ischemic infarction**—permanent damage caused by the rupture and thrombosis of a plaque. That's the type of stroke we're talking about next—rupture and thrombosis or embolism of a plaque that occludes arteries and kills the brain.

Stroke Syndromes by Artery

The goal of this section is to get you to think clinically and explore the lobes a bit further using the vascular territories. If you know the syndrome of a big artery—ACA, MCA, PCA—then when you are given symptoms that are part of the artery syndrome, you can anticipate what else to look for. Like STEMIs, the acute rupture and thrombosis to a brain artery happen in one artery, not multiple. Because it affects the brain, prior to any axonal decussation, all symptoms will be **contralateral to the lesion**. Because one lesion is affecting one area of the tract, **cortical strokes aren't bilateral**. And because the ACA perfuses some of the motor cortex (legs, feet) and the MCA separately perfuses the most of the motor cortex (everything else), you shouldn't have symptoms in the arms and legs. But because the axons travel through deep brain, you **CAN have symptoms in both the arms and legs** at the same time.

ACA stroke = lower extremity motor function and sensation loss. Because the ACA perfuses the medial aspect of both the motor cortex and primary somatosensory cortex (which control the lower extremities), a stroke of the ACA would compromise both motor function and sensation, but only in the lower extremities. The patient will have upper motor neuron lesion signs and symptoms, and, because of axonal decussation, they will be seen on the **contralateral side**.

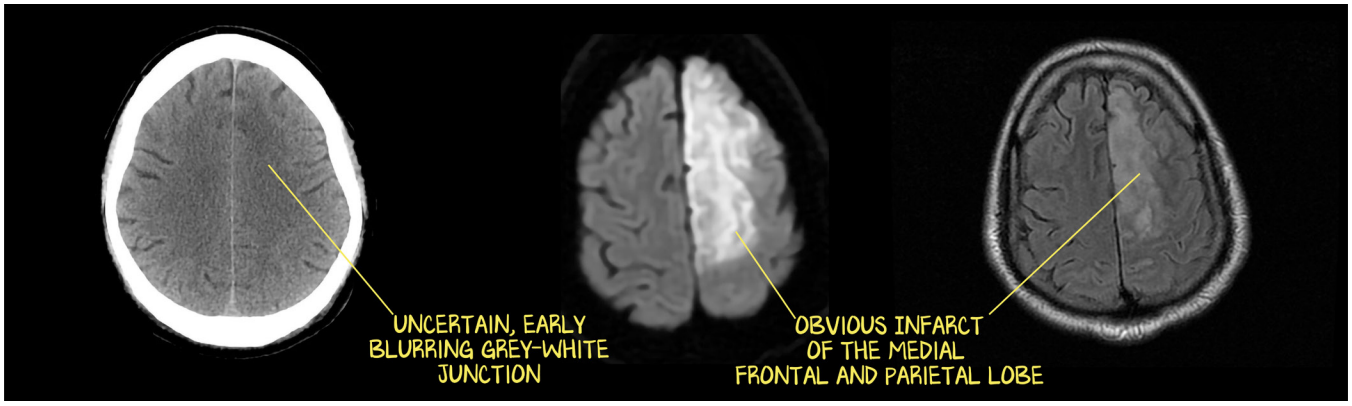

Figure 4.7: ACA Strokes on MRI
In each of the modalities—a noncontrasted CT on the left, a T2-weighted MRI in the middle, and a T1-weighted MRI on the right—there is an obvious difference between the damaged tissue and the healthy. The noncontrasted CT scan (on the left) isn't as clear as the other two. A noncontrasted head CT is done in minutes, performed to rule out hemorrhage, but can show evidence of ischemia. The MRIs take a long time to perform and aren't part of the initial assessment.

MCA stroke = upper extremity motor function and sensation loss. Because the MCA perfuses the lateral aspect of both the motor cortex and primary somatosensory cortex (which control the upper extremity and face), a stroke of the MCA would compromise both motor function and sensation in the upper extremities and face. This is why **facial droop** is a classic sign of stroke. The patient will have upper motor neuron lesion signs and symptoms, and, because of axonal decussation, they will be seen on the **contralateral side**.

However, the MCA covers a lot of territory. The MCA perfuses **Broca's area**, the loss of which would render the patient unable to form the words the brain is trying to say. Comprehension is intact, but the patient "can't get the words out." Writing, which would remain intact, is independent of vocalization. The MCA also perfuses **Wernicke's area** in the temporal lobe. This is the center required for fluent speech and speech comprehension. If lesioned, the patient will have normal cadence and the ability to form words well, but the words will be a string of incomprehensible nonsense. The MCA covers the top and front of the parietal lobe. MCA strokes cause **aphasia** (inability to speak) if in the dominant hemisphere (usually the left), or **hemineglect** (the patient ignores one half of the body) if in the nondominant hemisphere.

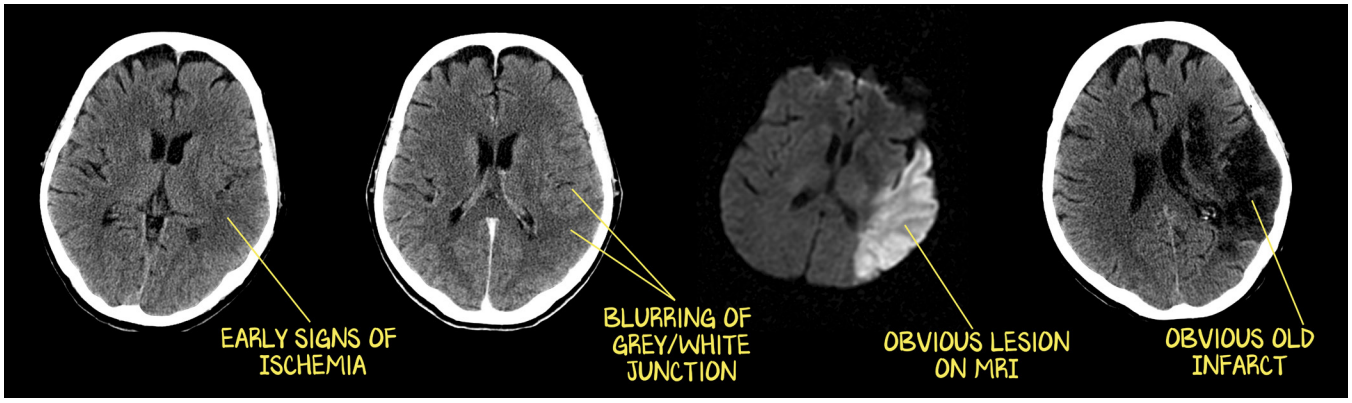

Figure 4.8: MCA Strokes on MRI
The changes of an untreated MCA stroke as they evolve over time. On the far left, there are early grey-white changes on this noncontrasted head CT. The second image is hours later, showing worsening of the grey-white blurring and vasogenic edema. The third is an MRI (it may look funny and fuzzy; it's a different view, but also the view that most obviously identifies the lesion) that confirms the suspicion of left MCA stroke (right of the image, left of the patient). Two weeks later, the consequence of the infarct, total annihilation of the grey-white junction with cavitation.

PCA stroke = contralateral homonymous hemianopsia with macular sparing. We haven't covered vision yet; that comes later in Special Senses. But you know that the motor and sensory nerves criss-cross, and that the occipital lobe does vision somehow. Contralateral means "the other side," homonymous means "the same for both eyes," and hemianopsia means "half (hemi-) the visual field is lost (-anopsia)." Visual hallucinations or other visual irregularities may also be present and can be explained by the fact that the PCA **irrigates the visual cortex.** The PCA also gives off an early branch to the **lateral thalamus**. The thalamus is the relay station for sensation, but not for motor function. Thus, the second key finding is **contralateral hemisensory loss** (any sensory finding will do) **without hemiplegia** (motor function is intact). The vision changes and loss of sensation with sparing of motor function are the two things you should look for.

Because there are vessels that perfuse the **parietal lobe** as well, there can be findings of parietal dysfunction. Whereas the MCA comes down from the front, the PCA comes up from the back. There is some overlap in symptoms. If the lesion is in the dominant hemisphere, it affects language, but it's not aphasia: it is instead **alexia** (inability to read) **without agraphia** (inability to write). If the **angular gyrus** is affected, there will be Gerstmann syndrome—finger agnosia (don't know their own fingers), acalculia (inability to count), and left-right confusion.

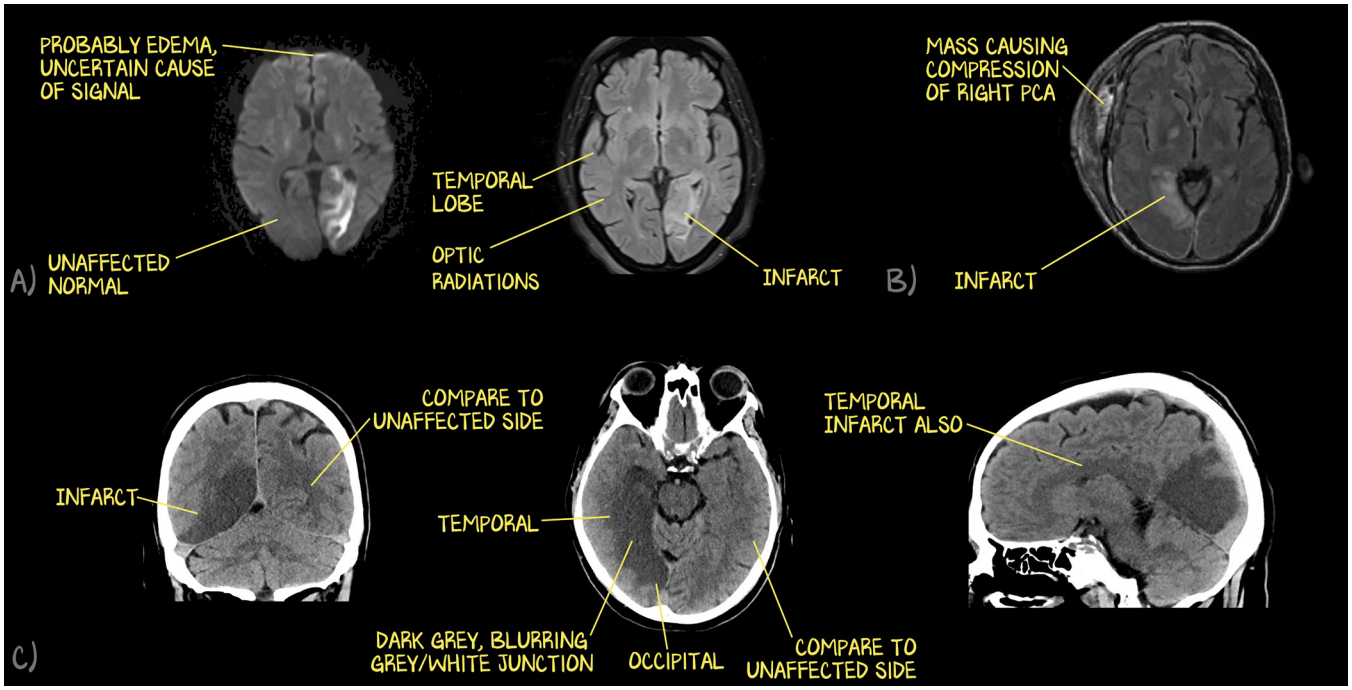

Figure 4.9: Radiological Patterns of Cerebral Circulation Strokes
More of the same things from the previous sections, except in the posterior circulation territory. (a) An infarct on FLAIR (left) and T1-weighted (right) MRIs, demonstrating how a positive signal can change depending on MRI settings. (b) A mass compressing on the right posterior cerebral artery (left of the image is the right of the patient). The outcome is ischemia, which looks the same as a distal artery thrombosis. (c) An old infarct (14 days), as seen on non-contrast CT. On the left and right images, the tentorium cerebelli delineates the inferior margin of the cerebral cortex and extenuates the dark grey lesion, which has obliterated the grey matter.

Lacunar Infarct Syndromes

There are two classic examples of lacunar infarcts leading to symptomatology. Recall that they usually occur in the deep brain. One is infarction of the internal capsule (lenticulostriate arteries, striatum for movement), and one is infarction of the thalamus (thalamogeniculate arteries, thalamo for sensory).

Lenticulostriate artery = pure motor function. The lenticulostriate arteries are branches of the MCA that perfuse the internal capsule. The most common presentation of a stroke in these arteries is a "**pure motor stroke**." There is no sensory deficit, and there is **hemiplegia**—both the upper and lower extremities are affected. This is how it will appear on your licensing exams. However, because both sensory and motor fibers run through the same posterior limb of the internal capsule, if there is sufficient vascular damage to provoke focal neurologic deficits, it is likely there will also be sensory deficits. Either **hemiplegia** (the "classic" pure motor stroke) or **hemiplegia** with **hemisensory loss**.

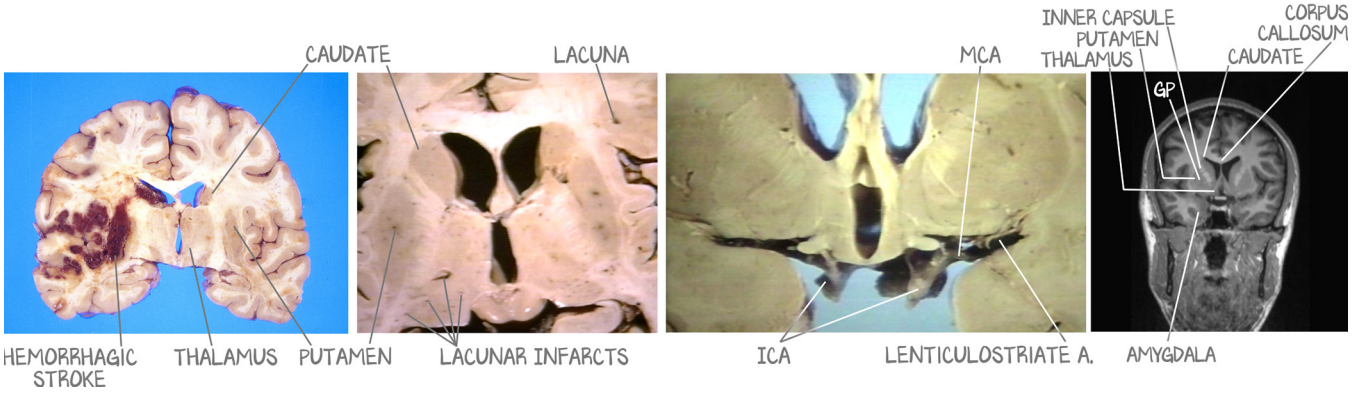

Figure 4.10: Lacunar Infarcts and Lenticulostriate Arteries
Gross coronal samples and a radiological image showing the various nuclei of the deep brain. In the gross samples, there are small holes in the white and grey matter. Those are lacunar infarcts. In the left-most sample, there is hemorrhaging within the internal capsule and putamen, the product of a lacunar infarct pathology—hyaline arteriolosclerosis secondary to hypertension.

Thalamogeniculate stroke = pure sensory. The thalamogeniculate arteries are penetrating arteries that feed the thalamus. If a lacunar infarct happens here, there will be the **contralateral loss of sensation**—pain, temperature, vibration, proprioception, touch—**without motor involvement**. This syndrome is always isolated to sensory, as the motor fibers never come near the thalamus.

Citations

Figures 4.2a, 4.2b, 4.2c, 4.3c, 4.4a, 4.4b, 4.4c, 4.5a, 4.5b, 4.5c, 4.6a, 4.6b, 4.6c: © Neurosurgical Atlas, Inc. www.neurosurgicalatlas.com.

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